Health Consultation

Evaluation of Contaminants: Domestic wells near Bainbridge Island Landfill (April 1996 - March 1998 Sampling data)

Kitsap County, Washington

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Prepared by
Washington State Department of Health
under cooperative agreement with the
Agency for Toxic Substances and Disease Registry

Washington State Department of Health

FOREWORD

The Washington State Department of Health (DOH) has prepared this health consultation under cooperative agreement with the Agency for Toxic Substances Disease Registry (ATSDR), an agency of the U.S. Public Health Service. The goal of the DOH and ATSDR is to identify and mitigate adverse human health effects resulting from exposure to hazardous substances in the environment. This report was prepared in accordance with methodologies and guidelines developed by ATSDR.

A health consultation provides advice on specific public health issues which may arise as a result of an actual or potential human exposure to a hazardous substance. Health consultations provide a means for DOH to respond quickly to a request for health information on hazardous substances and to make recommendations for actions to protect public health. DOH evaluates available information about hazardous substances at a site, determines whether exposures have occurred or could occur, and reports the potential harmful effects from exposure.

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BACKGROUND AND STATEMENT OF ISSUES

The Washington State Department of Ecology (Ecology) asked the Washington State Department of Health (DOH) to evaluate potential short-term health threats from vinyl chloride and other contaminants detected in domestic water supply wells in the vicinity of the Bainbridge Island Landfill in Kitsap County, Washington. This health consultation summarizes our evaluation of the public health implications resulting from actual or potential exposure to these contaminants, but does not attempt to identify the source(s) of contamination. Although Ecology is evaluating the Bainbridge Island Landfill (site) as a potential source of contamination, in this health consultation, the landfill is cited as a reference point only.

Ecology is currently overseeing a Remedial Investigation and Feasibility Study (RI/FS) for the site pursuant to the Model Toxics Control Act (MTCA). The site is located west of Eagle Harbor on Bainbridge Island and covers 40 acres, of which approximately 7 acres were used for refuse disposal. The landfill stopped accepting waste in 1975. The site comprises the northeast quarter of the northwest quarter of Section 33, Township 25 North, Range 2 East. Approximately 1,200 people live within 1 mile of the site and approximately 56,000 people live within 5 miles of the site (1990 census data).

As part of the Bainbridge Island Landfill RI/FS, Kitsap County is conducting quarterly sampling of 16 monitoring wells at the landfill and approximately 20 domestic water supply wells located in the vicinity of the landfill to evaluate water quality. DOH has been asked to review these data. This health consultation presents our findings.

METHODS

How DOH/OTS Evaluates Data

All monitoring well and domestic well sampling data were evaluated without regard to the source of contamination. Contaminants detected in drinking water wells exceeding a cancer and/or non-cancer health-based screening value were further evaluated in this health consultation. Screening values are media-specific concentrations used to select environmental contaminants for further evaluation. Contaminant concentrations at or below screening values are unlikely to pose a health threat. Contaminant concentrations exceeding screening values do not necessarily pose a health threat, but are further evaluated to determine whether they are at levels observed to cause toxic effects (referred to as toxic effect levels) in human population and/or laboratory animal studies. Exposure assumptions used in this health consultation are listed in Appendix A. Exposure formulas are listed in Appendix B.

Evaluating non-cancer risk:

To evaluate the potential for non-cancer adverse health effects as a result of exposure to contaminated environmental media (i.e, drinking water), a dose was estimated for each contaminant exceeding a health-based screening value. The doses were calculated for a scenario in which residents were assumed to be exposed to the maximum detected chemical

concentrations in their drinking water. The estimated dose for an adult and child through adulthood for each contaminant was then compared to ATSDR's minimal risk level (MRL) or EPA's oral reference dose (RfD). MRLs and RfDs are estimates of daily exposure of a human to a chemical that is likely to be without an appreciable non-cancer risk over a specified duration of exposure. They are derived from toxic effect levels obtained from human and laboratory animal studies. These toxic effect levels are expressed as either the lowest adverse effect level (LOAEL) or the no-observed adverse effect level (NOAEL). In human or animal studies, the LOAEL is the lowest dose at which an adverse effect is seen, while the NOAEL is the highest dose that did not result in any adverse health effects.

Because of the uncertainty associated with these data, the toxic effect levels are divided by safety factors (usually 100 or 1,000) to provide the more protective MRL or RfD. If a dose exceeds the MRL or RfD, the *potential* exists for adverse health effects. Thus, a dose only slightly exceeding the MRL or RfD would fall well below the toxic effect level. The higher the estimated dose is above the MRL or RfD, the closer it will be to the toxic effect level.

Evaluating cancer risk:

For screening of chemicals which are known or expected to cause cancer, it is assumed that no "safe" level exists, and EPA cancer slope factors are used to calculate an "estimated" cancer risk. An exposure which results in an estimated cancer risk of one additional cancer in a population of one million people exposed over a 70 year lifetime, is considered an acceptable risk, and is thus used as the screening value. In a population of one million men in the U.S., 500,000 are expected to develop cancer from all causes in their lifetime. For U.S. woman, the figure is 333,000. The additional estimated cancer risk means that if those one million men are exposed for 70 years to this level of the chemical, 500,001 will develop cancer. For those one million woman exposed, 333,001 will develop cancer.

How DOH Office of Toxic Substances evaluation methods differ from DOH Office of Drinking Water

Kitsap County Department of Public Works and the Bremerton/Kitsap County Health District have raised questions concerning the difference between the DOH Office of Toxic Substances (OTS) and DOH Office of Drinking Water (DW) in evaluating contaminants in drinking water supplies. Within the Environmental Health Division of DOH, both the DW office and OTS have roles in evaluating contaminants in domestic drinking water supplies. To assist the reader in understanding the differences between our respective roles and the criteria each of our offices use to evaluate exposure to contaminants (i.e., MCLs vs. ATSDR criteria), it is necessary to explain our respective mandates and authorities.

The DOH/DW Office regulates public water systems. In Kitsap County, responsibility for public systems serving 25 or fewer connections is delegated to the Bremerton-Kitsap County Health District. Federal and State drinking water standards, called *maximum contaminant levels (MCLs)* are used by the DOH/DW Office and Kitsap County Health District in regulating these

systems, and are *legally enforceable standards*. Although generally protective of public health, other non health-based considerations were made when developing MCLs, such as technological feasibility, implementation costs, and analytical limitations. In setting the MCL for vinyl chloride, for example, the Environmental Protection Agency has adopted a value which corresponds to the higher end of their acceptable risk range (one additional cancer per 10,000 people exposed, averaged over a lifetime). Also, the MCL does not consider routes of exposure other than ingestion (i.e., inhalation and dermal contact). For these reasons, DOH/OTS does not rely on MCL values alone as an indicator of potential health risk. As a result, DOH/OTS can, and sometimes does, *recommend* actions at levels below that which the DOH/DW Office may require of public water systems.

DOH/OTS's role is to provide technical assistance to agencies, groups, or individuals on actual or potential health risks from exposure to hazardous substances in the environment, such as contaminants in drinking water supplies. We evaluate data and draw conclusions about potential health risks based on *site-specific exposure scenarios* and by utilizing current chemical toxicity information and standard risk assessment methodologies to estimate exposures. In doing so, DOH/OTS goes a step further (beyond simply comparing the contaminant concentration to the numerical MCL value) in assessing potential health risk. Our conclusions can result in recommendations for actions to protect public health. However, our recommendations are just that, and are *not legally enforceable*.

DATA SUMMARY

Four of the domestic wells tested by Kitsap County are Group B wells (a public water system that serves more than one family but less than 25 people or 15 connections) and one is a Group A well (a public water system with 15 or more service connections or an average of 25 or more people per day for 60 or more days within a calendar year). The Group A well serves 1 residence and a school/day care facility. The remaining domestic wells are private wells serving individual residences.

Domestic supply wells and monitoring wells were initially analyzed for volatile organic compounds (VOCs), semi-volatile organic compounds (SVOCs), pesticides, herbicides, polychlorinated biphenyls (PCBs), total petroleum hydrocarbons (TPH), inorganics (metals), cyanide, and conventionals, such as total organic carbon, temperature, conductivity, etc. In later sampling rounds, fewer chemicals were tested, although VOCs continued to be analyzed. Domestic well sample analysis currently includes VOCs and conventional parameters (conductivity, nitrate, pH, alkalinity, chloride, total dissolved solids, dissolved oxygen, sulfate).

Some VOCs and SVOCs have been and continue to be detected at low levels in domestic wells (except for vinyl chloride, all VOCs and SVOCs are below ATSDR health-based screening values). Those compounds below ATSDR screening values do not pose a public health threat and will not be discussed in the remainder of the health consultation. Several inorganic compounds were also detected above comparison values in several of the domestic wells. Table 1 lists the domestic wells with the single highest chemical detections, and the range of detections

for that chemical in the well. The Table also lists each chemical's health-based screening value, the well types, well IDs, and approximate number of residences served by the well.

TABLE 1
CONTAMINANT RANGES AND MAXIMUM DETECTIONS
DOMESTIC WATER WELLS (AS OF MARCH 1998)

Chemical Name	Concentration Range (µg/l)	Carcinogenic Screening Value (µg/l)	Non-carcinogenic Screening Value (µg/l)	Well ID	Well Type	Number of Residences Served
1,1-Dichloroethane	ND - 0.25	NA	800 (MTCA B)	BOW37	Group B	6
1,1,1-Trichloroethane	ND - 0.2 (J)	NA	200 (MCL/LTHA)	BOW52	Private	1
1,2,4-TMB (pseudocumene)	ND - 0.3	NA	NA	BOW73	Group B	2
Acenaphthene	ND - 1.9	NA	600 (Child RMEG)	BOW31A	Private	1
Acetone	ND - 13	NA	1,000 (child RMEG)	BOW52	Private	1
Arsenic	***10.7 - 26.2***	***0.02 (CREG)***	***3 (child chronic EMEG)***	BOW31A	Private	1
Bis (2-ethylhexylphthalate)	2.7	3 (CREG)	200 (child RMEG)	BOW52	Private	1
Bromoform	ND - 0.1 (J)	4 (CREG)	200 (child RMEG)	BOW04	Private	2
2-Butanone (MEK)	ND - 6.9	NA	6,000 (child RMEG)	BOW52	Private	1
Carbon disulfide	ND - 0.4	NA	1,000 (child RMEG)	BOW15	Private	1
Chloroethane	ND - 0.2	NA	NA	BOW37	Group B	6
Chloroform	ND - 2.7	6 (CREG)	100 (child chronic EMEG)	BOW47	Private	2
Chloromethane	ND - 0.2 (J)	3 (LTHA)	3 (LTHA)	BOW45	Private	1
Chromium	ND - 9 (B)	NA	50 (child RMEG)	BOW64	Group A	1 + school
Dichlorodifluromethane	1.1 - 3.95	NA	2,000 (RMEG)	BOW37	Group B	6
Diethylphthalate	ND - 1.7	NA	8,000 (child RMEG)	BOW31A	Private	1

Lead	***1 (B) - 67***	NA	***15 (EPA Action level)***	BOW64	Group A	1 + school
Manganese	***589***	NA	***50 (RMEG)***	BOW12	Private	1
Methylmethacrolate	0.5	NA	640 (MTCA B)	BOW01	Group B	8
Methylene chloride	0.3	5 (CREG)	600 (child chronic EMEG)	BOW33	Private	1
Naphthalene	ND - 0.4 (J)	NA	200 (child inter. EMEG)	BOW35	Private	1
Toluene	ND - 0.4	NA	200 (child inter. EMEG)	BOW73	Group B	2
1,2,4-Trichlorobenzene	ND - 0.1 (J)	NA	70 (LTHA)	BOW35	Private	1
Trichlorofluromethane	ND - 0.9	NA	3,000 (child RMEG)	BOW56	Group B	4
Vinyl chloride	***0.3 - 0.77***	NA	***0.2 (child chronic EMEG)***	BOW37	Group B	6
Zinc	398 - 430	NA	3,000 (child chronic EMEG)	BOW01	Group B	8

µg/l = micrograms of chemical per liter of water (equals one part per billion).
RMEG = ATSDR's Reference Dose Media Evaluation Guide
MTCA B = WA Model Toxics Control Act Method B groundwater cleanup level
NA = Not available
MCL = Federal and state drinking water standard
CREG = ATSDR's Cancer Risk Evaluation Guide
LTHA = EPA's Lifetime Health Advisory for Drinking Water
EMEG = ATSDR's Environmental Media Evaluation Guide
EPA Action Level = EPA's action level for lead in drinking water
ND = not detected
J = estimated value between the calculated detection limit and reporting limit
B = estimated value less than the contract required detection limit, but greater than or equal to the method detection limit/instrument detection limit
bolded compounds = compounds exceeding an ATSDR screening value which required further evaluation

Discussion

After evaluating all of the sampling data, *DOH concluded that no health threat exists for people exposed for 1-5 years to any of the contaminants detected in the domestic wells to date. Although a very low chronic (long-term) health risk exists from exposure to the maximum concentration of individual contaminants (vinyl chloride in one well and arsenic in one well), there is no apparent public health hazard.* ATSDR uses the "*no apparent public health hazard*" category for sites where human exposure to contaminated media

is occurring or has occurred in the past, but the exposure is below a level of health hazard.

The Kitsap County Department of Public Works will continue sampling domestic supply wells quarterly through at least the end of 1998. DOH is working closely with the Bremerton-Kitsap County Health District and Ecology, and will continue to evaluate the sample results to determine future recommendations.

Contaminants exceeding a screening value which were further evaluated:

The following contaminants detected in individual domestic wells exceeded an ATSDR health-based screening value and were thus further evaluated in the health consultation:

- Arsenic
- Lead
- Manganese
- Vinyl chloride

In addition, 1,2,4-Trimethylbenzene (pseudocumene) and chloroethane (ethyl chloride) were detected, but have no published oral MRL's, RfD's, and/or slope factors against which estimated doses may be compared.

1,2,4-Trimethylbenzene is commonly found in paint thinner and is of low general toxicity. This compound is a general anesthetic at high concentrations, and at very high concentrations can cause central nervous system depression. Concentrations of 1,2,4-Trimethylbenzene found in the two drinking water samples were very low (0.2 µg/l and 0.1 µg/l), and due to their low toxicity would not be expected to cause adverse health effects.

Chloroethane was detected in one well (0.1 µg/l and 0.2 µg/l). Inhalation studies provide the only data in which to quantify cancer or non-cancer health effects for chloroethane. However, the estimated dose as a result of exposure to the maximum detected concentration of chloroethane in drinking water is well below the LOAEL and NOAEL for this compound, indicating that non-cancer health effects are not expected to occur. These health effects levels were derived from animal inhalation studies. A carcinogenicity assessment is currently under review by the EPA, thus a cancer risk cannot be derived.

Arsenic

Arsenic concentrations since 1996 have ranged from non-detect to 26.2 µg/l. The maximum arsenic detections, ranging from 10.7 µg/l (10/96) to 26.2 µg/l (9/96) were from well BOW31A, a private drinking water well located approximately 1,000 feet south of the landfill. This well has not been tested for inorganics since October 1996.

Arsenic is a naturally occurring element in the earth's crust and is widely distributed in the environment. Arsenic has been found naturally at higher levels in groundwater in some western

Washington locations. All humans are exposed to low levels of arsenic present in the environment. Both organic and inorganic forms of arsenic exist. Inorganic arsenic is generally more toxic than organic arsenic. Inorganic arsenic compounds are used to preserve wood and to make insecticides and weed killers. At high levels, arsenic can damage nerves, the stomach, intestines, and skin. Exposure to lower levels (300 to 30,000 ppb in food or water) can cause nausea, vomiting, diarrhea, decreased production of red and white blood cells, abnormal heart rhythm, blood vessel damage, and a painful sensation in the hands and feet. Perhaps the single most characteristic effect of long-term oral exposure to inorganic arsenic is a pattern of skin changes. This includes a darkening of the skin and the appearance of small "corns" or "warts" on the palms, soles, and torso. A small number of the corns may ultimately develop into skin cancer. EPA classifies arsenic as a known (class A) human carcinogen. Ingesting inorganic arsenic increases the risk of skin cancer and tumors of the bladder, kidney, liver, and lungs.

The estimated increased cancer risk due to chronic exposure to the maximum arsenic concentration is approximately four to five additional cancers per 10,000 persons exposed as an adult, and approximately six to seven additional cancers per 10,000 persons exposed as a child through adulthood (low increased cancer risk).

The MRL and RfD are based on a NOAEL of 0.0008 mg/kg/day observed in a large Taiwanese population chronically exposed to arsenic primarily from drinking water. The critical effects were keratoses and hyperpigmentation of the skin with possible vascular complications.

Although the estimated doses for a child exposed through adulthood and for an exposed adult at the site were just over the chronic oral MRL, they were below the LOAEL established for each systemic effect studied in another study (Tseng, 1977, Cebrian, 1983, Southwick, 1983). Several epidemiological studies of moderately-sized populations (20-200 people) exposed to arsenic in drinking water have detected no dermal or other effects at average chronic doses in the range of the estimated child and adult exposure doses for this well. As a result, the estimated doses for both the child exposed through adulthood and adult exposure scenarios were below levels expected to cause adverse non-cancer health effects.

Lead

Lead concentrations since 1996 have ranged from non-detect to 67 µg/l. The two maximum lead concentrations (67 µg/l and 25.6 µg/l, total and dissolved, respectively) were from well BOW64, a Group A public water system, located approximately 1,500 feet northeast of the landfill. This system serves approximately 45 students at a school and 1 residence. For all domestic wells sampled, these were the only 2 lead detections above the 15 µg/l federal action level. When this well was re-sampled the following month, lead concentrations were 1-2 µg/l, well below EPA's 15 µg/l action level.

Lead is a naturally occurring metal found in small amounts in the earth's crust. Most of it comes from human activities such as mining, manufacturing, the burning of fossil fuels, batteries, pipes, ammunition, and paint. Everyone has some lead in their bodies as a result of exposure to natural and anthropogenic (man-made) sources.

The non-cancer effects of lead are well known. At high doses, lead is toxic to the brain and can cause encephalopathy. Lower doses cause peripheral nervous system toxicity, kidney damage, blood disorders, and hearing impairment. The most sensitive toxic effect of lead poisoning is believed to be impaired development of the central nervous system in children. This effect has been measured by observing changes in the behavior of children, including performance in school. These changes have been measured at very low lead levels in the blood. The CDC has established a blood lead level of concern of 10 micrograms of lead per deciliter of blood (µg/dl). Children who have a blood lead level that exceeded this value are considered at risk and should have their exposure reduced. Fetal exposure from the mother to high levels of lead can cause premature birth and low birth weight.

Lead is classified by the EPA as a B2 (probable human) carcinogen. This classification was based on sufficient evidence of cancer in animals and inadequate evidence in humans. Several studies have demonstrated that high doses of lead in laboratory animals can cause kidney tumors. Quantitative carcinogenic analysis of lead in drinking water was not possible due to the lack of adequate studies from which to derive a cancer potency factor. The developmental effects of lead in children are recognized as the most sensitive toxic endpoint of lead exposure. As the non-cancer endpoint is the most sensitive indicator of exposure, the lack of cancer data is not significant in determining risk.

Long-term exposure to the maximum lead concentration detected in drinking water (67 µg/l) is not expected to increase blood lead levels in children above CDC's 10 µg/dl level of concern. EPA's Integrated Exposure Uptake Biokinetic Model (IEUBK-Version 0.99D), which incorporates a background blood lead level of approximately 4 µg/dl and assumes some lead exposure from soil, dust, air, and diet, indicates that blood lead levels in children between 1 and 6 years of age (those most susceptible) would not exceed 9 µg/dl at this level of exposure.

Manganese

Manganese concentrations since 1996 have ranged from non-detect to 589 µg/l. The maximum manganese detection (589 µg/l) was from well BOW12, a private well located approximately 600 feet east of the landfill.

Manganese is a widely distributed element that is essential for normal physiologic functioning in all animal species. Several health effects in humans have been associated with both deficiencies and excess intakes of manganese. Although there are many reports of toxicity to humans exposed to manganese by inhalation, much less is known about oral intakes resulting in toxicity. Only one limited study in primates by the oral route of exposure is available. While manganese

is clearly an essential element, it has also been demonstrated to be the causative agent in a syndrome of neurologic and psychiatric disorders that has been described in manganese miners. Other documented symptoms resulting from exposure to high doses of manganese include lethargy, increased muscle tonus, tremor and mental disturbances. The most severe symptoms were observed in elderly people, while children appeared to be unaffected. One case study suggests that for individuals with impaired liver function, intakes of manganese that would otherwise be safe may present a problem. In contrast to inhaled manganese, ingested manganese has rarely been associated with toxicity.

A study by Kondakis et al. (1989) raises some concern for possible adverse health effects (mild neurological signs) associated with a lifetime consumption of drinking water containing about 2,000 µg/L of manganese. A report by Kawamura et al. (1941) is the only epidemiologic study describing toxicologic responses in humans consuming large amounts of manganese dissolved in drinking water. In this study, the concentration of manganese at the time of exposure was estimated to be as high as 28,000 µg Mn/l. Another case study of manganese intoxication involved a 62-year-old male. The oral intake of manganese was estimated to be approximately equivalent to 40 mg Mn/day. These concentrations are well above the maximum concentration detected.

There is no human carcinogenicity data and inadequate animal carcinogenicity data for manganese. As a result, quantitative carcinogenic analysis of manganese in drinking water was not possible due to the lack of adequate studies from which to derive a cancer potency factor.

Although both the child through adulthood and adult exposure dose estimates were slightly above the chronic oral RfD, exposures were below levels demonstrated to cause toxic effect in the 1989 study described above (i.e, below the less serious LOAEL). Further, the study had substantial limitations and did not prove that chronic oral intake of manganese can lead to neurological changes in humans.

Vinyl Chloride

Vinyl chloride concentrations since 1996 have ranged from non-detect to 0.77 µg/l. Vinyl chloride has been detected at low levels in 12 of the 21 domestic wells sampled (through 6/98). The maximum concentration was 0.77 µg/l at well BOW37 in October 1996. This well is located approximately 800 feet northeast of the landfill. Vinyl chloride concentrations in this well over the subsequent 6 sampling rounds (April 1997-June 1998) dropped to 0.3-0.5 µg/l, and is currently 0.4 µg/l (6/98).

Vinyl chloride is a colorless gas at normal temperatures. All vinyl chloride is manufactured or results from the breakdown of manufactured substances, such as trichloroethylene, trichloroethane, and tetrachloroethylene (commonly used cleaning and degreasing compounds).

Most of the vinyl chloride produced in the United States is used to make polyvinyl chloride (PVC). PVC is used to make a variety of plastic products including pipes, wire, cable coatings and packaging materials. Other uses include furniture and automobile upholstery, wall coverings, housewares, and automotive parts.

Breathing high levels (>1,000 ppm) of vinyl chloride can cause dizziness and sleepiness. Animal studies have demonstrated that exposure to extremely high levels of vinyl chloride can damage the liver, lungs, and kidneys. Other animal studies suggest that long-term inhalation exposure to vinyl chloride may damage the sperm and testes and cause high blood pressure during pregnancy. Studies using pregnant animals show that breathing high levels (2-500 ppm) of vinyl chloride may harm their unborn offspring. Animal studies also show that vinyl chloride may cause increased numbers of miscarriages early in pregnancy. It may also cause decreased weight and delayed skeletal development in fetuses. The effects of drinking high levels of vinyl chloride are unknown. The MRL was derived from a LOAEL value of 0.018 mg/kg/day for an increased incidence of areas of cellular alteration in the livers of rats.

Results from several studies suggest that breathing air or drinking water containing low levels of vinyl chloride may increase the risk of developing cancer. Hepatic angiosarcomas in Sprague-Dawley rats were observed at doses approximately 3,000 times greater than doses estimated for persons chronically exposed to the highest vinyl chloride detection in well BOW37. Studies of workers who have been exposed to vinyl chloride over many years also indicate increased incidences of angiosarcoma of the liver. Brain, lung, and some blood cancers may also be attributed to chronic inhalation exposure to vinyl chloride. Studies of long-term exposure in rats indicate that increases in liver and mammary gland cancer may occur at very low levels of exposure in the air (5-250 ppm). The Department of Health and Human Services, International Agency for Research on Cancer, and EPA have determined that vinyl chloride is a human carcinogen.

EPA is currently reassessing vinyl chloride's carcinogenicity, and has thus removed the oral slope factor. However, for this health consultation, the former oral slope factor of 1.9 was used to estimate the excess cancer risk due to exposure to this compound. The estimated increased cancer risk, assuming chronic exposure to the maximum concentration of vinyl chloride in drinking water from well BOW37, is approximately 6 additional cancers per 100,000 persons exposed from childhood through adulthood, and approximately 4 additional cancers per 100,000 persons exposed as an adult (very low increased cancer risk).[1] The estimated doses for both the child through adult and adult exposure scenarios are below the less serious LOAEL for oral exposure, indicating that exposure to the maximum detected concentration of vinyl chloride is not expected to result in adverse non-cancer health effects.

[1] A review of Health District records indicate that BOW37 was initially drilled as a private well in 1976. County Assessor records indicate that homes were built and connected to the water supply in 1983, 1986, and the mid 1990s. As a result, estimated exposures, and thus risk, would be even less than this since a 30-year exposure duration was assumed for this health consultation.

Child Health and Developmental Effects

Arsenic:

Various epidemiological studies have reported an association between exposure to inorganic arsenic and increased risk of adverse developmental effects (congenital malformations, low birth weight, spontaneous abortions), both by the inhalation and oral routes of exposure. In these studies, however, other chemicals may have contributed to the observed effects. Animal studies, however, do support the view that arsenic is a developmental toxicant, causing a reduced birth weight, a variety of fetal malformations (skeletal and soft tissue), and increased fetal mortality. These data suggest that although inorganic arsenic is a developmental toxicant, the developing fetus is not especially susceptible, and teratogenicity or fetotoxicity are unlikely to be of concern except at doses that are also toxic to the pregnant female (i.e., doses several orders of magnitude greater than the doses estimated from exposure to arsenic from well BOW31A).

Although developmental effects resulting from the ingestion of arsenic in drinking water have not been extensively investigated, there is no convincing evidence that ingestion of arsenic, at least at the levels encountered in drinking water wells near the Bainbridge Island Landfill site, causes developmental toxicity in children.

Lead:

As indicated above, the most sensitive toxic effect of lead poisoning is believed to be impaired development of the central nervous system in children. It appears that some of the adverse health effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold.

Manganese:

It is recognized that neonates (from birth to 6 weeks) may be at increased risk of toxicity resulting from exposure to manganese because of a higher level of uptake from the GI tract and a decreased ability to excrete absorbed manganese. However, very little information exists on the developmental effects of manganese. The incidence of neurological disorders and the incidences of birth defects and stillbirths were elevated in a small population of people living on an island where there were rich manganese deposits. However, the lack of exposure data, the small sample sizes, and the absence of a suitable control group precludes associating these effects to manganese. The route of exposure was assumed to be primarily oral, but inhalation exposure was not ruled out. No studies were located regarding developmental effects in animals after oral exposure to manganese.

Vinyl chloride:

No human or animal studies were located regarding developmental or reproductive effects following oral exposure to vinyl chloride. However, some data suggests that fetuses, infants, and young children may be particularly susceptible to the toxic effects of vinyl chloride. Vinyl chloride can cross the placenta and enter the blood of fetuses. Developmental effects have been observed as a result of parental exposures to vinyl chloride in the air. A statistically significant increase in birth defects was observed in three cities in which facilities using vinyl chloride were located when compared to statewide and county wide averages. The greatest increases were malformations of the central nervous system, upper digestive tract, genital organs, and in the incidence of club foot.

Results of animal inhalation studies indicate that vinyl chloride produces developmental effects at concentrations that are also toxic to maternal animals. Maternal toxicity was evidenced by decreased food consumption, decreased body weight, and increased mortality. Delayed ossification was noted in fetuses at 500 ppm. Vinyl chloride exposed rats throughout gestation showed an increased incidence of hemorrhages, increased edema, decreased hemoglobin and leukocytes and decreased organ weights. However, doses at which developmental effects were observed were several orders of magnitude higher than estimated doses resulting from exposure to vinyl chloride from well BOW37.

Conclusions

No health threat exists for people exposed for 1-5 years to concentrations of contaminants detected in any of the domestic wells sampled to date.

Long-term exposure (DOH assumed 30 years) to the maximum detected vinyl chloride concentration (0.77 µg/l) poses a very low increased cancer risk. At current exposure levels, the risk is even lower. Conversely, the risk would increase if the concentration increased.

Long-term exposure to the most recent lead concentration (2.3 µg/l) in well BOW64 does not pose a health risk. However, because the previous lead concentration in this well was higher, only two rounds of metals analysis was conducted for this well, and this well serves a school, further evaluation of this well is appropriate.

Long-term exposure to the maximum detected arsenic concentration (26.2 µg/l) would pose a

low increased cancer risk. However, the concentration dropped to approximately one-third the maximum concentration in a follow-up sample which would pose an even lower risk.

Long-term exposure to the maximum detected manganese concentration does not pose a health risk.

Based on DOH's evaluation of all of the domestic well data provided to date, no apparent public health hazard exists as a result of exposure to contaminants detected in any of the wells.

Recommendations

1. Continue quarterly monitoring of domestic wells. Provide DOH with the results of the quarterly monitoring for review and evaluation.

2. Health education should be provided to residents who may still be concerned about exposures. DOH will work closely with the Bremerton/Kitsap County Health District to address local concerns.

3. Continue to monitor well BOW37 for VOCs to observe that vinyl chloride concentrations do not increase in subsequent quarters. If vinyl chloride (or other VOCs) show increasing trends or reach federal Safe Drinking Water Act Maximum Contaminant Levels (MCLs), exposures should be reduced or eliminated. DOH will continue to review and evaluate quarterly well monitoring results to determine future

recommendations; or reduce or eliminate exposure to contaminants from this well (options could include treatment or an alternate water source).❋

4. Well BOW31A should be re-tested by the owner to determine if the arsenic concentration continues to remain below a level of health concern (DOH can provide technical assistance and can evaluate test results should the well owner pursue testing).

5. Well BOW64 should be retested for lead (Pb) by the water system to observe whether the concentration remains below a level of health concern (i.e.- below EPA's 15 µg/l action level). Washington state drinking water regulatory monitoring requirements for Group A water systems should be adhered to.

6. Ecology's March 1995 and March 1998 letters recommending that the Bremerton/Kitsap County Health District limit its well site approval in the areas identified in the vicinity of the landfill should be adhered to.

Should future public health intervention become necessary, DOH will work with the appropriate agencies to address the possible long-term need for an alternate water source or treatment for wells determined to be at risk.

❋ Our recommendation to reduce or eliminate exposure to contaminants detected in this well is intended to prevent *future* exposures to potentially higher VOC concentrations from occurring in the event monitoring is discontinued. As previously indicated, since 1996, when this well was first tested, vinyl chloride levels have dropped in half and monitoring will continue through at least the end of 1999. DOH will continue to assess contaminant trends in this, and other domestic wells being sampled, to determine future recommendations.

Appendix A - Exposure assumptions:

For this health consultation, it is assumed that a completed exposure pathway exists and residents were exposed 350 days per year, for thirty years to the maximum detected chemical concentrations which exceeded a screening value (i.e., those contaminants highlighted in Table 1). This exposure duration accounts for potential past and future exposures, as well as current exposure. Both adult and child through adult exposure scenarios were evaluated. Adults were assumed to consume 2 liters of water per day, and children were assumed to consume 1 liter of water per day until 5 years of age and 2 liters of water per day for the remaining 25 years. Children were assumed to be exposed for 5 years at a 16 kilogram (kg) body weight, for 10 years at a 40 kg body weight, and for 15 years at a 70 kg body weight. Children and adults were assumed to be exposed to VOCs through ingestion (drinking) and non-ingestion (inhalation and dermal contact) routes. Non-ingestion exposures are assumed to occur during household activities such as cooking, bathing, and dishwashing.

Appendix B-Exposure formulas:

It is assumed that non-ingestion (inhalation and dermal) exposures are equal to exposures through ingestion.

Exposure dose = ((C x IR x EF x ED)/BW x AT)) x 2

Excess cancer risk = Exposure dose x CSF

where:
C = concentration of contaminant (μg/l)
IR = Ingestion rate (liters of water/day)
EF = Exposure frequency (days/year)
ED = exposure duration (total # of years in exposure period)
BW = body weight
AT = averaging time (70 years x 365 days/year)
CSF = Cancer slope factor (Estimates the excess upperbound lifetime probability of an individual developing cancer from an exposure)

References

1. Draft Remedial Investigation Report, Bainbridge Island Landfill, August 25, 1997. Kitsap County Department of Public Works, Solid Waste Division.

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3. Site visits, Bainbridge Island Landfill, April 24 and September 17, 1998.

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6. Integrated Risk Information System (IRIS), April 1998.

7. ATSDR Health Consultation Procedures, Agency for Toxic Substances and Disease Registry, May 1995.

8. Toxicological Profile for Vinyl Chloride, U.S. Department of Health and Human Services, Public Health Service, ATSDR,

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14. Centers for Disease Control and Prevention, National Center for Environmental Health. Childhood Lead Poisoning Prevention. Screening young children for lead poisoning: Guidance for State and Local Public Health Officials, November 1997.

15. American Cancer Society Homepage: 1998 Cancer statistics.

DEFINITIONS

EMEG: ATSDR's Environmental Media Evaluation Guide. A concentration in air, soil, or water (or other environmental media), which is derived from ATSDR's MRL, and below which adverse non-cancer health effects are not expected to occur. Separate EMEGs can be derived to account for acute, intermediate, or chronic exposure durations.

RMEG: ATSDR's Reference Dose Media Evaluation Guide. A concentration in air, soil, or water (or other environmental media), which is derived from EPA's RfD, and below which adverse non-cancer health effects are not expected to occur. RMEGs account only for chronic exposure.

MRL: ATSDR's Minimal Risk Level. An estimate of daily human exposure to a dose of chemical that is likely to be without an appreciable risk of adverse noncancerous health effects over a specified duration of exposure. MRLs are derived when reliable and sufficient data exist to identify the target organ(s) of effect or the most sensitive health effect(s) for a specific duration via a given route of exposure. MRLs can be derived for acute, intermediate, and chronic duration exposures by the inhalation and oral routes.

CREG: ATSDR's Cancer Risk Evaluation Guide. A concentration in air, water, or soil (or other environmental media), which is derived from EPA's cancer slope factor and carcinogenic risk of 10E-6 for oral exposure. It is the concentration that would be expected to cause no more than one excess cancer in a million persons exposed over a lifetime.

CHRONIC RfD: An estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure level of the human population, including sensitive subpopulations, to a potential hazard that is likely to be without an appreciable risk of deleterious effects (non-cancer) during a lifetime. It was developed to be protective for long-term exposure to a compound (7 years or longer).

CANCER SLOPE FACTOR: A plausible upperbound estimate of the probability of a response per unit intake of a chemical over a lifetime. The slope factor is used to estimate an upperbound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level of a potential carcinogen.

LOAEL: Lowest Observed Adverse Effect Level. LOAEL's have been classified into "less serious" or "serious" effects. In dose-response experiments, the lowest exposure level at which there are statistically or biologically significant increases in the frequency or severity of adverse effects between the exposed population and its appropriate control.

NOAEL: No Observed Adverse Effect Level. The dose of a chemical at which there were no statistically or biologically significant increases in frequency or severity of adverse effects seen between the exposed population and its appropriate control. Effects may be observed at this dose, but were judged not to be "adverse".

MCL: Federal Maximum Contaminant Level. A drinking water regulation established by the Safe Drinking Water Act. It is the maximum permissible concentration of a contaminant in water that is delivered to the free-flowing outlet of the ultimate user of a public water system. MCLs are enforceable standards.

CARCINOGEN: Any substance that can cause or contribute to the production of cancer.

CONTAMINANT: Any substance or material that enters a system (the environment, human body, food, etc.) where it is not normally found.

MONITORING WELLS: Wells developed to collect groundwater samples for the purpose of physical, chemical, or biological analysis to determine the amounts, types, and distribution of contaminants.

PLUME: An area of chemicals in a given media, such as groundwater.

REMEDIAL INVESTIGATION: A study designed to collect the data necessary to determine the nature and extent of contamination at a site.

COMPARISON VALUE: A concentration used to select contaminants of concern at hazardous waste sites that are further evaluated in the health assessment process. The terms comparison value and screening level are often used synonymously.

CLHA: Child Long-Term Health Advisory

MTCA: Model Toxics Control Act. Washington States hazardous waste cleanup law.

MCLG: Maximum Contaminant Level Goal.